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Delusional Misidentification Syndromes: *Separate Disorders or Unusual Presentations of Existing DSM-IV Categories?*

ABSTRACT

During the past 80 years, delusional misidentification syndromes (DMS), especially the Fregoli and Capgras syndromes, have posed challenges to mental health professionals due to a lack of comprehensive understanding of the syndromes and a lack of effective treatment. An issue that remains to be unresolved is whether DMS (either in its pure form or as embedded symptoms of other diagnoses) can be accommodated in the present *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV). During the past two decades, neurophysiological and neuroimaging studies have pointed to the presence of identifiable brain lesions, especially in the right frontoparietal and adjacent regions, in a considerable proportion of patients with DMS. Prior to the advent of such studies, DMS phenomena were explained predominantly from the psychodynamic point of view. Deficits in working memory due to abnormal brain function, are considered to play causative roles in DMS. In this article, we present two cases of Fregoli and Capgras syndromes and discuss the relevant theoretical and practical issues.

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Key Words: Fregoli, Capgras, delusion, misidentification, DMS

INTRODUCTION

Some sets of symptoms and signs exhibited by psychiatric patients can be challenging in terms of finding an appropriate fit within the current *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV). Some such signs and symptoms are those that make up delusional misidentification syndromes (DMS), which are related to dissociation and identification processes.[2] The DMS comprise signs and symptoms that are often viewed as part of other disorders, most commonly schizophrenia (paranoid type), affective disorders,[6] organic brain disorders (e.g., ischemic brain damage),[4] traumatic head injury,[5] and even typhoid fever.[3] As indicated by studies using abnormal event-related potentials, DMS may be related to working memory dysfunction affecting prefrontal, parietal, and adjacent brain regions.[1,2,4,5] The most common findings reported are lesions in the right hemisphere, specifically in the frontal, parietal, and adjacent regions.[1,2,4,5,13,17] Recently, brain imaging using single-photon emission computed tomography (SPECT) has been a valuable addition to investigate the pathophysiology of DMS.[16] Techniques designed to explore working memory function, such as the recording of event-related potentials, specifically P 300, have shown promise in the investigation of DMS, as some such studies have shown differences between DMS patients and controls.[18] The main DMS discussed in psychiatric literature are Capgras syndrome and Fregoli syndrome.[9]

In this article we provide a brief literature review, present two cases of Fregoli and Capgras syndromes, and discuss relevant issues.

CASE REPORTS

Case 1. Mr. P., a 59-year-old Caucasian man, came to our outpatient psychiatric clinic for follow-up treatment. He was previously diagnosed with bipolar disorder, mixed type, and had a history of multiple prior psychiatric hospitalizations and incarcerations. He had just been discharged from a day treatment program in which he had participated for a year and a half and was now deemed improved enough for outpatient clinic treatment.

Mr. P. reported that his past problems were related to his behavior, particularly when he "stalked women." He explained that he was doing relatively well until 15 years previously when he met a woman with whom he became romantically involved. He tried to "save" this woman (she was supposedly a prostitute and addicted to crack cocaine), which precipitated his decompensation. After his relationship with this woman ended, he became "different." He started following other women in the streets and would subsequently get arrested for harassment or hospitalized in inpatient psychiatric units. During one such hospitalization at another facility, the diagnosis of bipolar disorder was made. After each discharge or prison release, he would return to his original behavior and continue to follow women that he claimed he knew. He was eventually sent to a state psychiatric hospital where he stayed for over three years. After discharge from the hospital, he was placed in an assisted outpatient treatment program by court order, as he was deemed dangerous to the public.

During the many years of his stalking behavior, he was not known to be physically violent toward the women concerned, although he was once charged with attempting to hit a woman with his car. In this particular instance, Mr. P. claims he had no intention of hitting the woman with his car; rather, he simply drove close to where the woman was standing to initiate a conversation and offer her a lift.

He generally approached women in public places and would start inappropriate conversations focusing on their "future relationship" and the sexual aspects of it. When the women tried to get away from him, he would pursue them and even try to enter their homes in some cases. This led to several arrests, incarcerations, and hospitalizations.

Mr. P. was somewhat vague about details of his past behavior and tried to minimize his actions by blaming it on his "strange state of mind" during those times. He maintained that the past was irrelevant; what was more important, he maintained, was how well he did now with the help of treatment.

During his psychotherapy sessions, it became apparent that Mr. P. engaged in the repetitive stalking behavior because of his strong belief that each of the women he followed represented the same woman he met 15 years ago. This belief appeared fixed and resistant to change. Consistent with the paranoid quality of the syndrome, Mr. P. was often reluctant to reveal details regarding his delusion. When asked about this reluctance, he stated, "You never know who might be listening." His explanation as to why he would engage in the risky behavior repeatedly, knowing the consequences and having other means of satisfying his sexual desires, appeared to be a phenomenon fitting the description of Fregoli syndrome.

Mr. P. was raised by his father's younger brother and did not know who his biological parents were until he was 45. This, he felt, was very confusing for him and the resulting questions regarding his identity prevented him "from really getting well."

During his childhood, others perceived him as a "difficult child." He frequently got in trouble at school, but was able to graduate from high school in time. He was always very concerned about maintaining his health and refrained from smoking and using

drugs. He was also excessively concerned about his physical appearance.

Mr. P.'s biological father provided for him financially. After his father died, the family lawyer became Mr. P.'s legal custodian and managed the trust fund his parents left for him.

After his admission to the clinic, Mr. P. was continued on a combination of valproic acid and risperidone. He was also seen in once weekly individual therapy sessions. During the course of treatment, it became apparent that he was continuing his previous behavior of following women, despite knowing the risk of being arrested and hospitalized again. (He had stated repeatedly that "going back to jail or hospital is like death to me.") Mr. P.'s problems and treatment posed significant challenges for the treatment team. Diagnostic issues with his symptoms, his repetitive stalking behavior in spite of years of treatment, and potential for relapse remained serious concerns throughout his treatment at our outpatient facility. It was perplexing that the patient, who was a good-looking man and had secure financial resources, would continue to engage in behavior that exposed him to situations (jail and hospital) he dreaded.

Although Mr. P. initially denied that he was continuing to stalk women, the treatment team received reports from multiple sources that he was continuing to engage in the stalking behavior. Mr. P. was subsequently hospitalized, as he was deemed a threat to others.

Though Mr. P. had received a diagnosis of bipolar disorder from one or more previous treatment facilities, he did not exhibit signs of mania or major depression during the several months of treatment at our clinic, nor did he experience any hallucinations. He did, however, complain of troublesome anxiety from time to time.

At the time of this writing, Mr. P. was referred back to a day treatment program following this latest hospitalization.

Case 2. Ms. C., a 58-year-old woman, was brought to our psychiatric emergency room after she called the police and reported there was a stranger in her house. Ms. C. had a history of prior psychiatric hospitalizations and was previously diagnosed with schizophrenia (paranoid type). When the police arrived, she explained that her husband was not her husband but was a stranger. She became argumentative and combative toward the police officers. Due to her history of past psychiatric incidents involving the police, she was brought to the psychiatric emergency room. At the time of the incident, she was known to have consumed half a pint of brandy, and some of her symptoms were thought to be alcohol-related. When evaluated in the emergency room, she reported her distress was due to the impostor that had recently been substituted for her husband and that this impostor made her life miserable. She reported that she could not "take it anymore" and wanted to "get rid of him," so she called the police. She exhibited paranoid beliefs, such as the neighbors poisoning her, and reported auditory hallucinations. Her medical history and family history were non-contributory. She worked as a housekeeper but quit six months previously because she thought her employer was conspiring against her. On admission to the hospital, Ms. C. presented as well groomed with a somewhat agitated mood and labile affect, expressing paranoid ideation ("A woman down the street steals my belongings and substitutes it with old stuff.") and experiencing persecutory auditory hallucinations.

After admission, she was started on risperidone 2mg/day. Her paranoid symptoms improved. Her delusions of her husband being substituted by an impostor, however, persisted and did not appear to be related to her alcohol problems. While in the psychiatric unit, she also accused her attending physician of being substituted by an impostor. She was released after three weeks in the hospital with clinical improvement in psychotic symptoms, but the delusion that her husband was an imposter did not improve.

Her psychiatric status and present treatment involvement, if any, are unknown at the time of this writing.

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DISCUSSION

Fregoli syndrome is a disorder in which a person holds a delusional belief that different people are in fact a single person who changes his or her appearance or is in disguise. The condition is named after the Italian actor Leopoldo Fregoli, who was renowned for his ability to make quick changes in his appearance during his stage acts. It was first reported in a paper by Courbon and Fail in 1927. They discussed the case of a 27-year-old woman who believed she was being persecuted by two actors whom she often went to see at the

theatre. She believed that these people "pursued her closely, taking the form of people she knows or meets."[9]

Capgras syndrome is the delusion that an impostor has replaced a close friend or relative. It is named after Joseph Capgras (1873–1950), a French psychiatrist who first described the disorder in a paper he co-authored with Reboul-Lachaux in 1923. They used the term *l'illusion des sosies* (the illusion of doubles) to describe the case of a woman who complained that various "doubles" had taken the place of people she knew.[9]

Huang, Liu, and Yang reported in 1999 that of 364 patients admitted to a psychiatric hospital, nine fulfilled the criteria for Capgras syndrome. Four of these nine patients were proven to have abnormal anatomical lesions when they were studied using a combination of measures, such as electroencephalogram (EEG), brain computed tomography (CT), magnetic resonance imaging (MRI), and SPECT, and five cases were associated with "major physical illnesses."[19]

Feinberg and associates in 1999 reported the case of a 61-year-old man who suffered traumatic brain injury resulting in right frontal and left temporoparietal contusions and subsequently developed florid Fregoli-type DMS. This patient's "neuropsychological profile" closely resembled that of patients with Capgras syndrome previously reported in the literature. The authors concluded that a combination of executive and memory deficits may account for DMS associated with brain lesions.[20]

Edelstyn and Oyebode in a review of Capgras syndrome stated that even though DMS was traditionally considered to have its origins in psychodynamic conflict, recent studies have shown that between 25 and 40 percent of there cases are associated with organic disorders, which include dementia, head trauma, epilepsy, and cerebrovascular disease. They concluded that neuroimaging evidence revealed a link between Capgras syndrome and right hemisphere abnormalities, particularly in the frontal and temporal regions. They suggested that neuropsychological research provided empirical support for these findings by consistently reporting the presence of impairments in facial processing, which is considered a right hemispheric function. The authors opined that the study of Capgras syndrome will lead to a greater understanding of the neurological basis of psychotic experiences and may provide a paradigm for how the psychosis should be investigated.[21]

In a study of a patient suffering from Capgras syndrome using skin conductance response, Hirstein and Ramachandran found that the patient's Capgras delusion was "modality specific: he claimed that his parents were imposters when he was looking at them but not when speaking to them on the telephone." The authors stated that in such patients, the connections from face-processing areas of temporal lobe to the limbic system may be faulty and that far from being a "medical curiosity," Capgras syndrome may help explore the formation of new memories caught *in flagrante delicto*, a Latin term that means "in the act of committing the error."[22]

In 1996, Debruille and Stip examined the "evolution of hypothesis" regarding Capgras syndrome.[23] They reviewed over 60 studies published between 1866 and 1994 taking into account their relevance to clinical description and psychodynamic, neurological, and neuropsychological interpretations. The authors found that the earlier papers emphasized the psychodynamic point of view but the latter ones pointed to an organic dysfunction in a high percentage of cases. It is quite possible that the recent technological advances in neurophysiological and imaging studies contributed to this apparent shift.[23]

Fennig and associates described a 43-year-old patient who developed Capgras syndrome in association with a right frontal parasagittal meningioma. The delusion disappeared when the tumor was removed, pointing to the meningioma playing a causative role in the development of the delusion, in keeping with the assumption that right hemispheric lesions of the brain are often causative in the development of such delusions.[13]

Both of the cases we present here are examples of DMS encountered in clinical practice. DMS symptoms in some patients remit with the resolution of the underlying or associated illness, but in other cases, they remain unchanged even after the associated psychiatric illness

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remits.[7] The DMS component is more persistent than the accompanying psychosis in the schizophrenic patients with Capgras syndrome.[7] Relapse of the basic psychotic condition in the setting in which the syndrome had originally developed is often accompanied by reappearance of the syndrome.[7] A review of literature of the past 20 years dealing with treatment of DMS reveals that there are very few publications and no controlled studies that address the issue of effective treatment.

In a paper by Zanker, the author states the symptoms of DMS are very refractory to treatment despite various neuroleptic therapies.[12] Two reports on the possible effectiveness of pimozide, one in a patient who had failed to improve on haloperidol,[14,15] suggest that DMS may often be refractory to the commonly used neuroleptics. There is very little in the published literature regarding the effectiveness of atypical neuroleptics or SSRIs in the treatment of DMS. We found one report of the antidepressant mirtazapine being effective in a patient suffering from Capgras syndrome. The author speculated that this could be because mirtazapine may have antipsychotic properties.[11]

In a study published before the advent of atypical neuroleptics and SSRIs, DMS was reported to respond to various biological treatment methods. In the setting of depression, it may respond to tricyclic antidepressants.[8] In the setting of schizophrenia or "organic psychosis," it may respond to anti-psychotics. Specifically, in schizophrenic patients, DMS has a greater chance of responding to trifluoroperazine either given alone or in association with other psychotropics.[8] Treating the co-existing "organic dysfunction," if there is one, is equally important according to Christodolou.[8]

It is often unclear if DMS is a result of brain damage to specific areas of the brain, as some studies suggest,[1,2,4,5] or a manifestation of an underlying psychiatric disorder. It is also unclear if DMS has its rightful place as separate disorders or whether it should be considered part of existing DSM-IV categories. There is often a mismatch between such patients' symptoms and the DSM-IV categories and criteria. For example, the criteria for delusional disorders in DSM-IV would automatically exclude DMS.[10] According to DSM-IV, to be diagnosed with delusional disorder, the patient should exhibit only "one or more non-bizarre delusions in the absence of any other significant psychopathology." As the case reports presented here reveal, the delusions of these patients do have bizarre qualities, and these patients also had additional significant psychopathologies that qualified them for diagnoses of bipolar disorder and schizophrenia. In the case of Ms. C., other delusions and auditory hallucinations were evident during her hospitalization. Hence these patients' psychopathologies do not fit within the DSM-IV description of delusional disorders.

These patients' problems are of concern because of their extensive and continual involvement with the hospitals and mental health system. Our own awareness of the possibility that DMS may be caused by identifiable brain lesions came only after these patients were discharged. At this point, we performed an exhausting review of the available literature regarding the symptoms with which these patient presented. We found that DMS symptoms in patients often remain problematic in a waxing and waning manner, despite years of contact with mental health agencies. This suggests to us that the treatments available today are not fully effective for such patients. Furthermore, recent literature suggests that DMS may have a possible association with demonstrable brain lesions, and this seems to go largely unrecognized. In the available literature, a high proportion of patients with DMS phenomena have been noted to have identifiable brain lesions, but our patients did not have any documented brain scans or neurophysiological studies and were treated for their other diagnoses. This suggests that, in general, mental health professionals, including psychiatrists, seem to lack awareness regarding DMS and its possible causes.

There is a need to rule out identifiable brain lesions in patients suffering from psychotic

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phenomena, bipolar disorder, and symptoms of DMS, as this may be a subcategory of patients who require closer attention from a clinical and research perspective. Through such efforts we may also advance the field by answering the question of whether there should be an additional category or subcategory in the DSM for patients who exhibit DMS as their only psychiatric problem or as a phenomena in addition to other diagnoses. At present there is insufficient information to make such a determination. The information available today calls for a closer and comprehensive study of patients exhibiting DMS phenomena, and improvements in brain imaging and neurophysiological studies may play an increasingly prominent role in the evaluation of such patients.

CONCLUSION

The present state of knowledge regarding the DMS phenomena is still at a very incomplete and preliminary level. This calls for comprehensive evaluation of such patients aimed at better understanding of such phenomena from both the clinical and research perspectives. Neuroimaging procedures should be strongly considered in patients in whom the DMS phenomena remain resistant to treatment. More evidence has been accumulating that many of these patients may have identifiable neurological lesions involving the right cerebral hemisphere, some of which may even be treatable.[13] It is only through such efforts that a fuller understanding of such patients' problems and more effective treatment measures emerge. It is our hope that greater awareness of DMS as a whole and the possibility that DMS may be caused by neurological abnormalities will promote more studies in the future.

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